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TITLE PAGE

SUSPECTED AIR EMBOLISM THROUGH THE THORACIC VENTRAL INTERNAL VERTEBRAL VENOUS PLEXUS DURING HEMILAMINECTOMY IN DOGS

Running title: AIR EMBOLISM DURING SPINAL SURGERY

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ABSTRACT

This article describes a clinical observation made during thoracic spinal surgery in two dogs, leading to the suspicion of venous air embolism (VAE) through the ventral internal vertebral venous plexus.

Air bubbling from the pool of blood on the floor of the vertebral canal was observed in both cases accompanied with sudden cardiopulmonary disturbances – low end-tidal carbon dioxide pressure (PETCO₂), tachycardia and SpO₂ reduction. One case had dyspnoea as well. One case died. Similar observations have been made in human patients during spinal surgery, with fatal consequences in nearly 50% of cases.

Venous air embolism can spontaneously occur in dogs during thoracic spinal surgery and our aim is to raise awareness of this intra-operative, potentially fatal, complication.

Keywords: canine, air embolism, neurosurgery, spinal, ventral internal venous plexus.

INTRODUCTION

Intraoperative venous air embolism (VAE) is the entrainment of atmospheric air through lacerated veins or venous plexus in the operative field leading to circulatory disturbances or cardiac arrest (Mirski et al., 2007). This is possible when a pressure gradient exists between the veins at the surgical site and the heart (Palmon et al., 1997). Spontaneous intraoperative VAE is a life-threatening event, and a fatal outcome was reported for 10 out of 22 humans undergoing spinal surgery (Wills et al., 2005). It is also recognised during other surgeries in human patients (Mirski et al., 2007).

Venous air embolism has been associated with neurosurgery performed in the sitting position, and it is described during lumbar puncture, arthroscopy and hip arthroplasty (Mirski et al.,

2007). Air bubbling at the surgical site may be the first clinical indication of VAE in human patients (Wills et al., 2005).

In companion animals, iatrogenic VAE is reported to occur through intravenous catheters (Walsh et al., 2005; Holbrook et al., 2007; Pacifico et al., 2010; Boitout and Mahler, 2013; Mouser and Wilson, 2015), during pneumocystography (Ackerman et al., 1972, Thayer et al., 1980), laparoscopy (Gilroy and Anson, 1987), pharyngoscopy (Ober et al., 2006), oral surgery (Gunew et al., 2008) and cryosurgery (Harvey, 1978).

In this report we present two cases, both medium to large-breed dogs, where VAE was suspected during spinal surgery. Air bubbling at the surgical site was observed in both cases, concomitantly to signs of cardiopulmonary compromise in one case and death in another. The main differential diagnoses for the cardiopulmonary signs observed were excluded, although we could not obtain definitive of VAE evidence in either case.

CASE 1

An 8-years-old male neutered Dalmatian, weighing 29 kilograms, presented with a 2 day history of acute progressive, non-ambulatory paraparesis and the case was diagnosed with L2-L3 and L3-L4 intervertebral disc extrusion and T12-T13 disc protrusion on MRI (1.5 T Siemens Symphony Maestro Class).

The dog was pre-medicated with intravenous methadone (Comfortan; Dechra[®]) (0.3 mg/kg) and dexmedetomidine (Dexdomitor; OrionPharma[®]) (3.0 µg/kg); general anaesthesia was induced with alfaxalone intravenously (Alfaxan; Jurox[®]) (2.0 mg/kg), and maintained with isoflurane (Isoflo; Abbott[®]) in an oxygen and air mixture (1:1) delivered through a circle breathing system, allowing spontaneous breathing. Anaesthesia monitoring was performed by electrocardiography, pulse oximetry, capnography and non-invasive blood pressure measurement. Hartmann's solution was administered intravenously at 5 mL/kg/h.

Routine L2-L3 and L3-L4 left sided hemi-laminectomies were performed without complication. A left sided mini-hemilaminectomy and corpectomy were performed at T12-T13, where venous haemorrhage occurred from the ventral internal vertebral venous plexus and air bubbles were observed from the pool of blood on the floor of the vertebral canal.

Concomitantly, the dog showed marked inspiratory effort (evident as a deep inspiration), sudden sinusal tachycardia (increase from 75 to 125 beats per minute), and a decrease in partial pressure of expired end-tidal carbon dioxide (PETCO₂) (from 55 to 18 mmHg). Pulse oximetry values decreased from 95% to 93%. Respiratory rate, quality of peripheral pulses and blood pressure readings did not vary significantly. The endotracheal tube cuff and the capnograph were checked; two manual breaths were given by squeezing the circle system breathing bag. Thoracocentesis at the dorsal half of the eighth intercostal space of the left hemithorax was performed with no retrieval of air. These manoeuvres ruled out endotracheal cuff leakage, capnograph failure and pneumothorax as a cause for the decrease in PETCO₂.

Whilst a cause for the decreased PETCO₂ was sought, the surgical site was flushed with 20 mL boluses of saline (and again up to 100 mL) and the solution disappeared rapidly as if 'suctioned' into the vertebral canal. It was suspected that saline and air were aspirated through the lacerated ventral internal vertebral venous plexus into the systemic circulation. The mini-hemi-laminectomy site was packed with gelatine sponge (Spongostan, Ethicon[®]) to prevent more air entrainment. Subsequently, the heart rate and ventilation parameters stabilised. The surgical site was closed routinely.

A post-operative computed tomography scan of the thorax (Siemens Somatom Emotion 16, Erlangen, Germany) did not show evidence of significant pneumothorax or pleural fluid. There was a very small amount of free air in the caudal aspect of the left pleural space that was suspected to be associated with the intra-operative thoracocentesis.

Arterial blood gas analysis was within normal limits when sampled post-operatively.

The dog recovered well from anaesthesia, was ambulatory the following day and remained normal 7 months later.

CASE2

A 13-years-old male neutered Collie cross dog, weighing 14.4 kilograms, presented with a 2 week history of progressive back pain and paraparesis. He had a 2 years history of an asymptomatic, grade 4 out of 6 systolic heart murmur. His vital parameters were within normal limits and no arrhythmias were detected on clinical examination. Complete haematology was normal. Biochemistry was unremarkable except from mild increases in cholesterol: 7.99 mmol/L (reference range 2 – 7 mmol/L) and ALKP 248 U/L (reference less than 220 U/L). These changes could be related to corticosteroid treatment, prescribed by the referring veterinarian 2 weeks before presentation. We considered this patient as ASA physical status classification of 3.

The dog underwent general anaesthesia to perform an MRI scan (1.5 T Siemens Magnetom Essenza). The anaesthetic protocol consisted of acepromazine (ACP 2mg/mL, Novartis[®]) (14 µg/kg) and butorphanol (Torbugesic, Zoetis[®]) (0.2 mg/kg), followed by intravenous propofol to effect (PropoFlo Plus; Zoetis[®]) and inhaled isoflurane (Isoflo; Abbott[®]) at 2% in 1:1 oxygen and air. The patient remained stable during anaesthesia and he was diagnosed with a T12-T13 intervertebral disc herniation.

The following day the dog was anaesthetised to perform a left-sided hemilaminectomy at T12-T13. He was pre-medicated with acepromazine (ACP 2mg/mL, Novartis[®]) (7 µg/kg) and methadone (Comfortan; Dechra[®]) (0.2 mg/kg) intravenously. Anaesthesia was induced with propofol intravenously (PropoFlo Plus; Zoetis[®]) (1 mg/kg), and maintained with inhaled isoflurane (Isoflo; Abbott[®]) at 2% in 1:1 oxygen and air mixture, allowing spontaneous breathing through a circle breathing system. He received Hartmann's solution at 3.0 mL/kg/h.

A reaction was observed on positioning of the towel clamps, therefore a bolus of fentanyl (Fentadon; Dechra[®]) (1.5 µg/kg) followed by continuous rate infusion of fentanyl (Fentadon; Dechra[®]) (0.2 µg/kg/minute) and ketamine (Narketan; Vetoquinol[®]) (20.0 µg/kg/minute) was given. Anaesthesia monitoring was performed by electrocardiography, pulse oximetry, capnography and non-invasive blood pressure measurement. Heart rate was 60 beats per minute, respiratory rate 10 respirations per minute and mean arterial blood pressure 70 mmHg for the first 40 minutes of surgery. At that stage, suddenly, the heart rate increased to 150 beats per minute but the rhythm remained sinus, the systolic blood pressure increased by 30 mmHg and the respiratory rate remained unchanged. An acepromazine bolus (2mg/mL, Novartis[®]) (28 µg/kg) was given. The mean arterial blood pressure dropped to 48 mmHg as well as the PETCO₂ from 52 mmHg to 30 mmHg, and the SpO₂ from 100% to 95% but the tachycardia persisted. Around 15 minutes after these observed anaesthetic changes (55 minutes into the surgery), air bubbles were also seen coming from the haemorrhagic pool formed in the floor of the opened vertebral canal. At that time, the heart rate was still high (150 beats per minute) and the mean arterial blood pressure was still low (52 mmHg) with a PETCO₂ of 34 mmHg and SpO₂ of 100%. The dog was started on positive pressure ventilation at a frequency of 12 breaths per minute, with a tidal volume of 130 mL and peak inspiratory pressure of 12 cmH₂O and received intravenous colloid solution at 5 mL/kg/h. The surgeons flooded the surgical field with 20 mL and applied pressure for 5-10 minutes several times to try and stop bleeding from the vertebral sinus. Blood gas analysis performed around the time the surgeon witnessed the bubbles, revealed a reduced PaO₂ of 248 mmHg (a PaO₂ in the region of 600 mmHg would be expected based on the high inspired fraction of oxygen) and marked hypercapnia with accompanying acidosis (PaCO₂ of 76.8 mmHg, pH 7.146, HCO₃ 26.5, BE -2). Subsequently, the heart rate dropped to 40 beats per minute and the patient went into cardiopulmonary arrest. Open chest resuscitation was unsuccessful. No

blood was found in the thoracic cavity while entering the thorax, ruling out haemothorax as the cause of the cardiac arrest. It was also observed that the left lung did not expand completely, possibly due to positioning of the ET tube or bronchial obstruction or perforation. Post-mortem examination ruled out iatrogenic perforating trauma to the thorax from the surgical wound. No pulmonary lesions were found to explain why the left lung did not expand during resuscitation. The heart did not show macroscopic lesions on routine post mortem examination. Unfortunately, the heart was not submerged in water prior to opening the right ventricle.

DISCUSSION

Venous air embolism is a recognised complication in humans when the surgical field is above the level of the heart, creating a negative pressure gradient between the veins at the surgical site and the right atrium (Palmon et al., 1997). This allows air to be suctioned into a damaged open vein (Mirski et al., 2007). The greater the distance between the surgical site and the heart, the greater the pressure gradient (Palmon et al., 1997). Large amounts of air can enter the venous system with a pressure gradient as small as 5.0 cmH₂O (Albin, 2011). Neurosurgery in the sitting position in humans is associated with the highest incidences of VAE - 19% in a recent study (Ganslandt et al., 2013) but others have found that this complication can occur with the patient in prone or lateral position (Albin, 2011).

In a dog undergoing thoracic surgery in sternal position, the ventral internal vertebral venous plexus is ~10-20 cm above the heart (increasing with larger breeds), creating a negative pressure gradient and therefore a risk for VAE, especially in a large deep chested dog.

Negative pressure in the thoracic cavity during spontaneous inspiration decreases the intravascular pressure allowing air entrainment through cancellous bone or peripheral venous plexuses (McCarthy et al., 1990). In this situation air bubbles may be visible with fluctuations

of intrathoracic pressure associated with expiration (Wills et al., 2005). In mechanically ventilated patients, the positive intrathoracic pressure during inspiration could reduce the risk of VAE.

The severity of the VAE depends on the volume and rate of air embolization. With large and rapid emboli, immediate cardiovascular collapse can occur due to right ventricular outflow obstruction (Durant et al., 1947) or increased right ventricular afterload, hypotension and myocardial ischemia (Geissler et al., 1997). Smaller volumes of air in the pulmonary vascular bed may stimulate endothelin-1 receptors leading to pulmonary vasoconstriction exacerbating ischemia (Tanus-Santos et al., 2000).

Dyspnoea, low PETCO₂ and low oxygen saturation commonly occur. This provokes a deep inspiration that maximises the pressure gradient between the ventral internal vertebral venous plexus and the right atrium, allowing more air entrainment which can be fatal (Adornato et al., 1978).

Intraoperative bubbling of air may be the first sign of VAE during spinal surgery and could be key for early recognition (Wills et al. 2005). This is however different from the relatively common observation during hemilaminectomy of decrease and increase in venous haemorrhage at the site of an open ventral vertebral venous plexus (and within the cancellous bone) during inspiration and expiration, due to normal fluctuations in intra-thoracic pressure. Electrocardiogram changes include tachyarrhythmia, depression of the S-T segment (Geissler et al., 1997) and peaking in P wave in the early stages. Other clinical signs include auscultation of a mill-wheel murmur, decreasing PETCO₂ and hypotension (Adornato et al., 1978). Air bubbling at the surgical site, decreases in PETCO₂, oxygen saturation and blood pressure were observed in both cases. Inspiratory effort (evident as a deep inspiration) was also observed in case 1.

Precordial doppler ultrasound and transoesophageal echocardiography are sensitive non-invasive techniques used in humans to monitor for the appearance of VAE (Mirski et al., 2007). Venous air embolism can be confirmed on CT angiogram, thoracic radiographs or post-mortem examination as the presence of gas or frothy blood in the right atrium or blood vessels (Albin et al., 1991) (Mouser and Wilson, 2015). This is however a rare observation (Geissler et al., 1997) and cardiac collapse can occur with no intra-cardiac air visible (Adornato et al., 1978). In case 2, the heart was not submerged in water prior to opening the right ventricle to look for the presence of air bubbles but this is something pathologists should be aware of when performing heart examination in situations where VAE is suspected. Calculating physiological dead space by use of the Bohr-Enghoff equation would be an elegant way of showing a sudden increase in dead space in the cases presented. However, several factors impeded us in doing so: (i) in case 1 we did not obtain an arterial blood gas measurement during the event, but only post-operatively when the situation had already been stabilised; (ii) in both cases no baseline arterial blood gas measurements were performed so we would not be able to show a potential change in physiological dead space; (iii) in both cases the PETCO₂ was measured and not the mixed expired PCO₂ which is needed to calculate dead space using the Bohr-Enghoff equation; and (iv) the tidal volumes of both dogs before and during the critical events were not measured, which limit the use of the Bohr-Enghoff equation. Finally, any cause of pulmonary hypoperfusion would result in an increase in physiological dead space. An increase in physiological dead space is therefore not diagnostic for a VAE and would therefore not have provided additional evidence towards a diagnosis of VAE.

In cases of VAE, general recommendations include stopping nitrous oxygen when given (as this gas expands the volume of the embolus) and providing 100% oxygen, flooding or packing the surgical site to prevent more air entrainment (Wills et al., 2005), fluid-therapy to

maintain central venous pressure (Jadik et al., 2009) and lowering the operative site below the level of the heart to decrease the magnitude of the pressure gradient (Palmon et al., 1997). Re-positioning the patient has been shown not to be beneficial in dogs when moved from dorsal to lateral recumbency (Mehlhorn et al., 1994, Geissler et al., 1997). Positive pressure ventilation has been suggested as a preventive measure (Jadik et al., 2009): it increases intrathoracic pressure and therefore central venous pressure during the inspiratory phase of respiration, and stops reflex spontaneous inspiration after VAE, reducing the probability of a fatal bolus of air being suctioned into open veins (Adornato et al., 1978). High positive end-expiratory pressure (PEEP between 5-10 mmHg, i.e. 6.8-13.6 cmH₂O) during positive pressure ventilation has also been proposed in humans to prevent VAE (Jadik et al., 2009) but its use is controversial (Mirski et al., 2007). Giebler et al. suggested that it does not reduce the incidence of VAE and it worsens the cardiovascular performance (Giebler et al., 1998), and Perkins and Bedford suggested in 1984 that it could increase the risk of paradoxical embolism in patients with patent oval foramen (Perkins and Bedford, 1984), although these references apply to humans. Aspiration of air from the right ventricle is performed in people through a specifically developed multi-orifice central line aspiration catheter (Bunegin-Albin Air Aspiration Catheter Cook, Inc., Bloomington, IN), positioned at the junction of the vena cava and right atrium (Wills et al., 2005).

Conclusion

Venous air embolism can occur in dogs undergoing thoracic spinal surgery, although it was not possible to confirm the presence of air in the cardiovascular system in our cases. We based our diagnosis on the observation of air bubbling in the surgical field and subsequent haemodynamic and respiratory changes in case 1 and cardiac arrest in case 2. Such findings, along with compatible respiratory and cardiac changes, should alert surgeons and

anaesthetists to the possibility of VAE during spinal surgery in dogs and this needs to be considered as a serious, potentially fatal, intra-operative complication. Packing the surgical site with haemostatic sponges and active communication between the surgeon and the anaesthetist should take place to implement corrective measures.

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